6. Multiple viruses can be generated at the same time since plaque purification is unnecessary.

Dated: December 13, 2002.

Jack Spiegel,

Director, Division of Technology Development and Transfer, Office of Technology Transfer, National Institutes of Health.

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BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Government-Owned Inventions; Availability for Licensing

AGENCY: National Institutes of Health, Public Health Service, DHHS.

ACTION: Notice.

SUMMARY: The inventions listed below are owned by agencies of the U.S. government and are available for licensing in the U.S. in accordance with 35 U.S.C. 207 to achieve expeditious commercialization of results of federally-funded research and development. Foreign patent applications are filed on selected inventions to extend market coverage for companies and may also be available for licensing.

ADDRESSES: Licensing information and copies of the U.S. patent applications listed below may be obtained by writing to the indicated licensing contact at the Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804; telephone: 301/496–7057; fax: 301/402–0220. A signed Confidential Disclosure Agreement will be required to receive copies of the patent applications.

Immunotherapy With In Vitro-Selected Antigen-Specific Lymphocytes After Nonmyeloablative Lymphodepleting Chemotherapy

Mark E. Dudley, Steven A. Rosenberg, John R. Wunderlich (NCI)
DHHS Reference No. E–275–2002/0–US–01 filed 06 Sep 2002
Licening Contact: Jonathan Dixon; 301/435–5559; *dixonj@od.nih.gov.*

This invention discloses a novel method of treating cancer. The approach uses autologous T-cells, which are selected for their highly avid recognition of an antigen expressed by the cancer. In studies performed at the National Cancer Institute (NCI), this method has proven effective in promoting the regression of cancer in patients with metastatic melanoma.

The treatment of 13 patients at NCI resulted in tumor shrinkage of at least 50 percent in six of the 13, and several patients remain cancer free more than a year after treatment. All of the patients enrolled in this trial had been unresponsive to previous therapies including, surgery, radiation and chemotherapy. This method represents a step forward in the treatment of cancer and offers a clinically proven approach to effectively promote the regression of tumors. Not only may this method apply to a variety of cancers, but it may also be applicable in treating other diseases such as AIDS, immunodeficiency, or other autoimmunity for which immune effector cells can impact the clinical outcome.

Humanized Anti-TAG 72 CC49 for Diagnosis and Therapy of Human Carcinomas

Syed V. Kashmiri (NCI), Jeffrey Schlom (NCI), Eduardo Padlan (NIDDK)
DHHS Reference No. E–013–2002/0–US–01 filed 28 Jun 2002
Licensing Contact: Jonathan Dixon; 301/435–5559; *dixonj@od.nih.gov*

Tumor associated glycoprotein (TAG–72) is expressed on the cells of a majority of human carcinomas, including colorectal, gastric, pancreatic, breast, lung, and ovarian. The murine monoclonal antibody (mAb) CC49 specifically recognizes TAG–72 and has a higher affinity for TAG–72 than its predecessor, B72.3.

The present invention discloses new humanized variants of CC49 that have a higher binding affinity to TAG–72 than previous humanized variants. Identified as HuCC49V15 and HuCC49V14, these variants also retain low immunogenicity of variable regions using sera of patients vaccinated with murine CC49.

These variants have potential benefits for use in the detection and/or treatment of a range of human carcinomas. Certain fields of use may not be available. Please contact OTT for information regarding the availability of specific fields of use.

Identification of Potential Ovarian Cancer Tumor Markers and Therapeutic Targets

Dr. Amir Jazaeri *et al.* (NCI)
DHHS Reference No. E–310–2001/0–US–01 filed 13 Feb 2002
Licensing Contact: Catherine Joyce; 301/435–5031; *joycec@od.nih.gov*

Genes that are differentially expressed in cancerous ovarian tissue as compared to normal ovarian tissue were identified using microarray technology. This technique was used to characterize gene expression patterns in BRCA–1 associated tumors, BRCA–2 associated tumors, sporadic tumors and immortalized ''normal'' ovarian epithelial cells. As a result of this analysis, genes that are up-regulated in ovarian cancer were identified. Approximately two-thirds of the sequences identified were previously known genes, while approximately one-third were expressed sequence tags (ESTs), representing sequences that are cloned and identified but not yet characterized. Eighty-three (83) genes were over-expressed in 50% of all tumors and these over-expressed sequences may be used as markers for ovarian cancer and/or targets for therapy.

The above-mentioned invention is available for licensing on an exclusive or non-exclusive basis.

A Metastasis Suppressor Gene on Human Chromosome 8 and Its Use in the Diagnosis, Prognosis, and Treatment of Cancer

Naoki Nihei (NIEHS), J. Carl Barrett (NCI), Natalay Kouprina (NCI), Vladimir Larionov (NCI)
DHHS Reference No. E–238–2001/0–US–01 filed 21 Dec 2001
Licensing Contact: Matthew Kiser; 301/435–5236; *kiserm@od.nih.gov*

The subject technology is directed to a gene on human chromosome 8 that suppresses metastasis of prostate cancer. The gene has been shown to suppress the metastatic ability of rat prostate cancer and is down-regulated in human prostate cancers from metastatic foci. Embodiments of the technology include gene therapy to prevent the metastasis of human cancer, in particular prostate cancer, use of the gene as a clinical marker in the diagnosis and prognosis of cancer, in particular prostate cancer, and the development of small molecules that mimic the effect of the gene product.

The present invention provides an isolated or purified nucleic acid molecule consisting essentially of a nucleotide sequence encoding the metastasis suppressor gene located at p21–p12 on human chromosome 8, which has been named Tey 1, or a fragment thereof comprising at least 455 contiguous nucleotides.

Detection and Quantification of Cripto-1 in Human Milk Using ELISA

Caterina Bianco, David S. Salomon (NCI)
DHHS Reference Nos. E–290–2000/0–US–01 filed 26 Jan 2001 and E–290–2000/0–PCT–02 filed 23 Jan 2002 (PCT/US02/02225)
Licensing Contact: Brenda Hefti; 301/435–4632; *heftib@od.nih.gov*

Cripto-1 (CR1) is a member of the epidermal growth factor (EGF)-related families of peptides and is involved in the development and progression of various human carcinomas. In particular, CR1 overexpression has been detected in 50–90% of carcinomas of the colon, pancreas, stomach, gallbladder, breast, lung, endometrium and cervix. Current methodologies of cancer detection, *e.g.* immunohistochemistry, can be time consuming, inconvenient and oftentimes, inaccurate, and therefore, a need exists for more efficient, reliable and less time consuming methods of detection. The invention relates to such a method of detection. The inventors disclose methods for the detection and quantification of CR1 in human milk, using an ELISA-based protocol. Thus, this test could be used to more effectively detect and perhaps stage cancers. Additionally, should particular tumor cells, *e.g.* breast tumor cells, express a sufficiently high level of CR1, it may be possible to use the disclosed assay to detect and measure CR1 in human serum and/or plasma. Claims to these routes of detection are also present in the patent application. As such, a novel, efficient and useful in vitro diagnostic and prognostic test is now available to suitable commercial partners.

Improving Chemotherapy by Increased Killing of Tumor Cells and Protection of Normal Cells Through p38 Kinase Inhibition

Dmitry Bulavin and Albert J. Fornace, Jr. (NCI)

DHHS Reference Nos. E–235–2000/0–US–01 filed 07 Nov 2000 and E–235–2000/0–PCT–02 filed 06 Nov 2001 (PCT/US01/47669)

Licensing Contact: Catherine Joyce; 301/435–5031; *joycec@od.nih.gov*

Responses to genotoxic stress include the initiation of cell-cycle arrest and the maintenance of cell-cycle arrest during DNA repair. Although maintenance of G2/M checkpoints is known to involve Chk1, Chk2/Rad53 and upstream components, the mechanisms involved in initiation of the G2/M checkpoint are less well defined. The inventors have discovered that p38 kinase has a critical role in the initiation of a G2/M delay after genotoxic stress such as ultraviolet radiation. The inventors contemplate that p38 MAPK inhibition will enhance the efficacy of chemotherapy by inhibiting the initiation of G2/M arrest in stressed cells and promoting the progression of such cells into M phase.

The above-mentioned invention is available for licensing on an exclusive or non-exclusive basis.

Pyrimidine Phosphorylase as a Target for Imaging and Therapy

RW Klecker and JM Collins (FDA)

DHHS Reference Nos. E–156–1999/0–US–01 filed 19 Jan 2001 and E–156–1999/0–PCT–02 filed 18 Jan 2002 (PCT/US02/01216)

Licensing Contact: Brenda Hefti; 301/435–4632; *heftib@od.nih.gov*

The present invention describes methods to diagnose and monitor the treatment of tumors with high expression of thymidine phosphorylase (TP). Overexpression of TP has been shown to correlate with angiogenesis, and this fact can be used, via TP's enzyme function, to preferentially label angiogenic cells through the introduction of relevant precursors. These precursors consist of labeled thymine analogues which are converted by TP into retained cell-components. This can allow for the non-invasive imaging of tumors with high angiogenic activity. The technique can also be used to kill tumor cells by providing the analogues in higher concentrations or with therapeutic isotopes so as to be toxic to cells with high TP levels.

Dated: December 13, 2002.

Jack Spiegel,

Director, Division of Technology Development and Transfer, Office of Technology Transfer, National Institutes of Health.

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Tryptophan as a Functional Replacement for ADP-ribose-arginine in Recombinant Proteins

Dr. Joel Moss *et al.* (NHLBI), DHHS Reference No. E–160–2002/0–US–01 filed 28 Jun 2002 Licensing Contact: Marlene Shinn; 301/435–4426; *shinnm@od.nih.gov.*

Bacterial toxins such as cholera toxin and diphtheria toxin catalyze the ADP-ribosylation of important cellular target proteins in their human hosts, thereby, as in the case of cholera toxin, irreversibly activating adenylate cyclase. In this reaction, the toxin transfers the ADP-ribose moiety of Nicotinamide Adenine Dinucleotide (NAD) to an acceptor amino acid in a protein or peptide. ADP-ribosylation leads to a peptide/protein with altered biochemical or pharmacological properties. Mammalians proteins catalyze reactions similar to the bacterial toxins. The ADP-ribosylated proteins represent useful pharmacological agents, however, their use is limited by the inherent instability of the ADP-ribose-protein linkage.

The NIH announces a new technology wherein recombinant proteins are created that substitute phenylalanine or tryptophan for an arginine, thereby making the protein more stable, and better suited as agents for therapeutic purposes. The modification creates an effect similar to ADP-ribosylation of the arginine. An example of a protein that can be modified is the defensin molecule, which is a broad-spectrum antimicrobial that acts against infectious agents and plays an important role in the innate immune defense in vertebrates.

Identification of Anti-HIV Compounds Inhibiting Virus Assembly and Binding of Nucleocapsid Protein to Nucleic Acid

Drs. Robert Shoemaker and Michael Currens (STB, DTP, DCTD, NCI), Drs. Alan Rein and Ya-Xiong Feng (DRP, CCR, NCI), Drs. Robert Fisher, Andrew Stephen, Shizuko Sei, Bruce Crise, and Louis Henderson, and Ms. Karen Worthy (SAIC-Frederick), DHHS Reference No. E–121–2002/0 filed 08 Oct 2002, Licensing Contact: Sally Hu; 301/435–5606; *hus@od.nih.gov*

This invention identified potent inhibitors of HIV particle assembly and nucleocapsid/nucleic acid binding. Two series of active antiviral compounds are described in this invention. One series